Amendments to the Specification:

Please replace the paragraph beginning at page 11, line 24 with the following amended paragraph:

The preparation of polyclonal antibodies is well-known to those skilled in the art (e.g., Green *et al.*, *Production of Polyclonal Antisera*, In: Immunochemical Protocols (Manson, ed.), pages 1-5 (Humana Press 1992) and Coligan *et al.*, *Production of Polyclonal Antisera in Rabbits, Rats, Mice and Hamsters*, In: Current Protocols in Immunology, section 2.4.1 (1992)). In addition, various techniques common in the immunology arts can be used to purify and/or concentrate polyclonal antibodies, as well as monoclonal antibodies (Coligan, *et al.*, Unit 9, Current Protocols in Immunology, Wiley Interscience, 1994).

Please replace the paragraph beginning at page 12, line 16 with the following amended paragraph:

In addition, methods of *in vitro* and *in vivo* multiplication of monoclonal antibodies are well-known to those skilled in the art. Multiplication *in vitro* can be carried out in suitable culture media such as Dulbecco's Modified Eagle Medium (MEM) or RPMI 1640 medium, optionally replenished by mammalian serum such as fetal calf serum, or trace elements and growth-sustaining supplements such as normal mouse peritoneal exudate cells, spleen cells, and bone marrow macrophages. Production *in vitro* provides relatively pure antibody preparations and allows scale-up to yield large amounts of the desired antibodies. Large scale hybridoma cultivation can be carried out by homogenous suspension culture in an airlift reactor, in a continuous stirrer reactor, or in immobilized or entrapped cell culture. Multiplication *in vivo* may be carried out by injecting cell clones into mammals histocompatible with the parent cells (e.g., ~~osyngeneic~~ syngeneic mice) to cause growth of antibody-producing tumors. Optionally, the animals are primed with a hydrocarbon, especially oils such as pristane (tetramethylpentadecane) prior to injection. After one to three weeks, the desired monoclonal antibody is recovered from the body fluid of the animal.

Please replace the paragraph beginning at page 14, line 12 with the following amended paragraph:

The effects of intact pathogenic *Mycoplasma hyopneumoniae*, nonpathogenic *M. hyopneumoniae*, and *M. flocculare* on intracellular free Ca^{2+} concentrations ($[Ca^{2+}]_i$) in porcine ciliated tracheal epithelial cells were determined. Briefly, the ciliated epithelial cells had basal $[Ca^{2+}]_i$ of 103 ± 3 nM (n = 217 cells). The $[Ca^{2+}]_i$ increased by 250 ± 19 nM (n = 47 cells) from the basal level within 100 seconds of addition of pathogenic *M. hyopneumoniae* strain 91-3 (300 μg/mL), which lasted about 60 seconds. In contrast, nonpathogenic *M. hyopneumoniae* and *M. flocculare* at 300 μg/mL failed to increase $[Ca^{2+}]_i$. In Ca^{2+}-free medium, pathogenic *M. hyopneumoniae* still increased $[Ca^{2+}]_i$ in tracheal cells. Pretreatment with thapsigargin (1 μM, 30 minutes), which depleted Ca^{2+} store in the endoplasmic reticulum, abolished the effect of *M. ~~hyoneumoniae~~ hyopneumoniae*. Pretreatment with pertussis toxin (100 ng/mL, 3 hours) or U-73122 (2 μM, 100 seconds), an inhibitor of phospholipase C, also abolished the effect of *M. hyopneumoniae*. The administration of Mastoparan 7, an activator of pertussis toxin-sensitive-protein ($G_{i/o}$), increased $[Ca^{2+}]_i$ in ciliated tracheal cells. These results suggest that pathogenic *M. hyopneumoniae* activates receptors that are coupled to $G_{i/o}$, which in turn activates a phospholipase C pathway, thereby releasing Ca^{2+} from the endoplasmic reticulum. Thus, Ca^{2+} serves as a signal for the pathogenesis of *M. hyopneumoniae*.